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A SCREENING LEVEL RISK ASSESSMENT OF THE INDIRECT IMPACTS FROM THE COLUMBUS WASTE TO ENERGY FACILITY IN COLUMBUS, OHIO

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ABSTRACT

Testing for emissions of dioxins from the stack of the Columbus, Ohio Waste to Energy (WTE) municipal solid waste combustion facility in 1992 implied that dioxin concentrations in stack gas averaged 328 ng TEQ/m^3. The incinerator has been in operation since the early 1980s. In mid-1994, US EPA's Office of Research and Development (ORD) and EPA's Region 5 Office collaborated on a screening risk assessment to evaluate the potential indirect impacts of these emissions. This paper describes this assessment. The exposure setting is a hypothetical dairy farm where individuals on the farm obtain their beef, milk, and vegetables from home sources. A 70-year exposure scenario is considered, which includes 45 years of facility operation, with MACT coming on-line 15 years after the incinerator has been operating, followed by 25 years of impact due to residual soil concentrations. Soil dermal contact, inhalation, and breast milk exposures were also considered for this assessment. The "source" term, or dioxin loadings to this setting, were derived from air dispersion modeling of emissions from the Columbus WTE. A key finding of the assessment was that exposures to dioxin in beef and milk dominated the estimated risks, with excess cancer risk from these two pathways estimated at $2.8*10^{-4}$. A second key finding was that over 90% of a lifetime of impact from these two pathways, and the inhalation and vegetable ingestion pathways, has already occurred due to pre-MACT emissions.

INTRODUCTION

The purpose of this exercise is to make a screening level estimate of the possible indirect exposures and excess cancer risks resulting from emissions of dioxin-like compounds from the Columbus Incinerator. This analysis was conducted by ORD at the request and with the assistance of EPA's Region 5, and used by the Region in regulatory activities regarding this facility. Further information on the use of this assessment by Region V can be obtained from the EPA Region V Public Affairs Office.

The analysis is based on an exposure scenario involving individuals who obtain 100% of their vegetables, beef and milk from a hypothetical subsistence farm. Other exposure pathways considered include breast milk ingestion, a childhood pattern of soil ingestion, soil dermal contact, and inhalation. The concentrations of dioxin toxic equivalents (abbreviated hereafter as TEQs) in soil, vegetables, beef and milk are estimated from modeled ambient air concentrations. In order to determine this air concentration, 9 actual Grade A Dairy Farms in Franklin County were located for EPA by the Ohio EPA. The air concentrations at these nine farms were modeled and then averaged to represent the air concentration at the hypothetical exposure setting. Therefore, the exposure media concentrations, and particularly the foods in this assessment, beef, milk, and vegetables, could be interpreted as averages for local conditions. In other words, the 100% contact rate for individuals at a single subsistence farm could instead be interpreted as individuals obtaining 100% of their food for ingestion (or 100% of air for inhalation, etc.) from local sources. In general, the strategy was to devise a conservative yet feasible scenario so that the screening exercise could lead to an exposure and risk estimate that was within the realm of possibility but not likely to be exceeded in the actual population.

This qualification is a judgement on the part of the authors and would need to be verified with more site specific information on local food production and consumption. A screening assessment is intentionally conservative because a principal use of such an assessment is to determine if further evaluations are necessary. Secondly, with a lack of site-specific knowledge, it is possible that other exposure patterns could exist or other exposure assumptions may be warranted which would increase exposure estimates above those from the initially conservative scenarios. For example, higher exposure and risk estimates could have been made for the Columbus site by: 1) assuming an individual's lifetime dose is the sum of the dose received during breast feeding, the dose due to a pattern of childhood soil ingestion, and doses due to adult patterns of inhalation, ingestion, and so on; the current approach is to discuss breast feeding impacts and childhood soil ingestion impacts separately from adult exposures; 2) considering ingestion of other food types which might be produced in the area (i.e., other dairy products, chickens, eggs, pork), 3) using air levels at specific farms rather than an average of nine farms, 4) by considering accidental releases, fugitive emissions, upsets, and accidents (only average emission levels based on emission testing was considered), and so on. It was judged that combining some of these other conservative assumptions with the key assumption of 100% of vegetables, beef and milk impacted may produce exposure estimates that would be considered unreasonably high.

The air-to-soil and air-to-biota models are extensively described in the draft dioxin exposure reassessment document (1). The algorithms and the justification for assignment of model parameters are described in that document and not repeated here. Also, validation exercises described in that document generally found that the air-to-biota models realistically predict concentrations in these media.

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GENERAL STRUCTURE OF THE ASSESSMENT

The setting of this scenario is a dairy farm. Individuals residing on this farm obtain their daily complement of beef and milk from animals raised on the farm. Also, the farm has a home garden where the family's daily ingestion of vegetables are grown. A pattern of childhood soil ingestion is evaluated for this site, as is a pattern of soil dermal contact based on agricultural activities (tilling and other field work). Breast milk ingestion is considered for this site. Finally, inhalation impacts are also evaluated.

Exposures are to be assessed for a 70-year period of exposure corresponding to an assumed 45-year operation of the incinerator followed by 25 years of residual soil impacts. Four time “periods” are modeled:

- **Period 1**:   The incinerator was assumed to start operations on January 1, 1982. The capacity of the incinerator is 2000 t/day, and records indicate that the average municipal solid waste handled was 1600 t/day. Of 6 furnaces and boilers available, on average 4.22 were in use on a continual basis.   Stack tests conducted in 1992 indicated that the concentration of dioxins in the stack emissions equalled 328 ng TEQ/m^3, and that the total emission of dioxins from 4.22 boilers was $3.1*10^{-5}$ g TEQ/sec. It was assumed that these emissions were representative of emissions from 1982 to mid-1994, the 12.5 years of Period 1. In 1994, combustion improvements were made which were designed to reduce dioxin emissions.
- **Period 2**: Stack tests conducted in 1994 indicated that these combustion improvements reduced TEQ emissions by about 73%. This reduction resulted from a reduction in stack gas emissions coupled with a reduced concentration in those stack gases. Assuming the same 4.22 boilers on line, the emission for Period 2 was assumed to be $8.4*10^{-6}$ g/sec.   This reduced rate of emissions was assumed to occur for a 2.5 year period from July 1, 1994 until January 1, 1997 when Maximum

Achievable Control Technology (MACT) will be assumed to be in place.

- **Period 3**: MACT will be assumed to reduce emissions by 99% from what they were in Period 2, leading to an emission rate during that time of $8.4*10^{-8}$ g/sec. Also, it will be assumed that the facility operates for 30 additional years, which has the incinerator then operating for a total of 45 years until 12/31/26.
- **Period 4**: Impacts for the final 25 years of a 70-year scenario are based on residual soil concentrations. The 70-year duration of exposure assumes individuals that are born in 1982 and reside in this location for 70 years.

Air dispersion modeling runs corresponding to the period of start-up until combustion improvements in 1994 were conducted by the Ohio EPA (2). In that study, the Ohio EPA used the ISCLT2 model and estimated direct inhalation impacts from the Columbus incinerator. EPA Region 5 conducted further ISCLT2 model runs corresponding to the time after combustion improvements were assumed to be on-line in 1994 until the incinerator was assumed to shut down on 12/31/26. The Region 5 modeling study incorporated the same basic modeling methodology as was used by the Ohio EPA, and further details on the ISCLT2 application can be found in OEPA (2). The dispersion modeling runs supplied annual average "air concentration/stack emissions", or χ/Q, estimates for points surrounding the facility. The χ/Q values have units of [$\mu g/m^3$]/[g/s] and are defined as an average annual predicted ambient air concentration per unit emission rate. The Ohio EPA focused on the "maximally exposed individual" in their inhalation risk assessment (2). This individual was located about 0.6 miles away and the χ/Q was 0.055. For the purpose of this indirect screening study, nine licensed Grade A dairy farm sites in Franklin County were identified by the Ohio EPA. These farms were located between 5 and 12 miles away, and the χ/Q for the nearest of these farms was 0.0081, with the average being 0.0036. Air concentrations for this screening assessment, in units of $\mu g/m^3$, are then estimated as the product of the emission rate of TEQs, in g/s, times this average χ/Q of 0.0036. These air concentrations were then used in food chain models to determine resulting TEQ concentrations in soils, plants (including vegetables and cattle forages/grains), and animal food products (milk, beef).

Deposition in this screening assessment was calculated as the airborne concentration of particle bound TEQs (in units of pg TEQ/m^3) times a deposition velocity (in units of cm/sec; appropriate conversions made so that depositions are in proper units of g/m^2-yr). The dry deposition velocity assumed was 0.2 cm/sec. This was the velocity of deposition found by Koester and Hites (3) for dioxins depositing in two sites in Indiana. Further, it was assumed that annual wet deposition is equal to annual dry deposition. Koester and Hites (3) also found relatively equivalent amounts of annual dry and wet deposition in their monitoring study.

The stack emission modeling framework is depicted in Figure 1. Full details of this approach are given in the dioxin exposure reassessment document (1) and not repeated here. Briefly, total emissions are apportioned into a vapor and a particle phase. Vapor phase dioxins are assumed to transfer to vegetation which cattle consume. Two vegetation categories include pasture grass and non-grass vegetation, such as hay, silage, or grain. Particle depositions impact these vegetation reservoirs, and particle depositions are also used to calculate soil concentrations. A representative cattle diet for calculation of beef concentrations includes 48% pasture grass, 48% non-pasture grass vegetation, and 4% soil. A lactating cattle diet assumes less time pasturing: 90% nonpasture grass vegetation, 8% pasture grass, and 2% soil. The concentration in the fat of beef or milk is calculated as the weighted average concentration in the diets of the cattle times a lipid-based bioconcentration factor. This bioconcentration factor was developed based on experiments on a lactating cow as described in McLachlan, et al. (4).

The dioxin exposure document discusses the impact of feedlot fattening on beef concentrations. Although no data exists, the available literature suggests that feedlot fattening reduces concentrations of dioxins in fat by one-half compared to concentrations upon entry into the feedlot. The dioxin document assumes a one-half reduction factor in an air-to-beef model validation exercise described in the dioxin exposure reassessment document (1). This screening exercise, however, conservatively assumes that the home slaughtered cattle do not go through a period of feedlot fattening prior to slaughter. The implication of this assumption is further discussed in the Results Section below.

Figure 2 shows the modeling approach for estimating impacts from soil contamination. This approach was used to estimate impacts for the fourth time period. The modeling is essentially similar to the modeling of the impact of incinerator emissions with two exceptions. One, the source of air-borne vapor and particle phase dioxins is the soil and not a distant incinerator stack. Volatilization releases vapor phase dioxins and wind erosion suspends particle bound dioxins. Two, soil impacts from deposition is not considered. Rather, the soil concentration resulting from 45 years of depositions is the starting point for impact estimation during Period 4. The reservoir at that time is assumed to dissipate at a rate corresponding to a 10-year half-life.

Impacts to vegetables, beef, and milk are assumed to reach steady state immediately in each of the four periods that are considered in this assessment. This is a simplification, although steady state between air and vegetation is expected to reach steady state in a matter of days or weeks assuming the air concentrations remains steady, and a steady state is expected to be reached in the milk of lactating cows within about two months given a steady ingestion rate of dioxins (5). While steady state is not expected to be reached in the tissue of non-lactating cows, McLachlan (6) notes that cattle are most often slaughtered while they are relatively young and their body fat pool is still expanding. Therefore, measured beef fat concentrations tend to mirror that in milk fat (6).

The breast milk model used in this screening assessment assumes that the lipid based concentration in the breast milk is the same as the lipid based concentration in the maternal tissue. A steady state level of dioxins in lipids is assumed to be reached in the mother instantly during each of the four periods. This was done as a simplifying assumption for purposes of describing the differences that could occur from long term exposure to different levels of combustor emissions. Assuming steady state in the mother before it would be reached has the net effect of overpredicting the concentration of dioxins in maternal tissue and hence also in the mother’s milk.

The mother’s milk algorithm also assumes that the dioxin concentrations remain constant in the mother’s milk while she is breast feeding. In reality, breast milk concentrations are likely to be declining due to declines in maternal levels because of accelerated elimination caused by breast feeding. Sullivan et al (7) estimated that the steady state assumption may lead to a 20% overestimate in dose to the infant. In order to estimate the concentration in mother's milk, the steady state dose received by the mother will be assumed to be the dose from consumption of beef and milk during each of the 4 periods. Further details on the breast milk exposure pathway can be found in the dioxin exposure reassessment document (1).

Like mother's milk, soil also will not reach steady state quickly given constant depositions of dioxins. The soil concentration when the incinerator begins emitting in 1982 is assumed to be zero. Dioxins build up in the soil over time given a particle deposition rate and an assumption of a half-life of 10 years in the soil. The strategy for determining the impacts to soils over time is qualitatively described in an example as follows. The soil concentration at the end of year 2 is a function of the soil concentration at the end of year 1, plus all that accumulated during year 2, minus any dissipations of the concentration left over from year 1 and the dissipation of residues depositing during year 2. This procedure, translated mathematically, led to calculation of unique soil concentrations for each of the four periods which reflected unique depositions for the first three periods, and no further depositions but only dissipation during the fourth period.

The above paragraphs have described how exposure media concentrations are calculated during each of the four periods. For the 70-year exposure and risk estimates presented, time-weighted average concentrations are determined. For example, the modeled air concentrations and years in each of the four periods are: 0.11 pg TEQ/m^3 and 12.5 years for Period 1, 0.03 and 2.5, 0.0003 and 30, 0.000007 and 25. Therefore, the time-weighted concentration of TEQs in air is calculated as:

$$\underline{(0.11 * 12.5) + (0.03 * 2.5) + (0.0003 * 30) + (0.000007 * 25)} \qquad \textbf{(1)}$$

which equals 0.02 pg TEQ/m^3.

For most pathways, exposures are assumed to occur for the full lifetime, with exposure media concentrations calculated in this manner. Two of the exposure pathways considered, however, are not 70-year exposures. One is a childhood pattern of soil ingestion, which has a duration of 5 years only. The other is a breast milk exposure pattern, which is assumed to occur during the first year of an individual's life. These exposure pathways will be evaluated for each of the four time periods. In other words, it will be assumed for purposes of this screening assessment, that infants are exposed via breast milk in each of the four periods, and that children ingest soil for 5 years during each of the four periods. A 5-year evaluation of a soil ingestion pattern is technically incorrect for the second incinerator period since the second period is only 2.5 years long, not five years. It is estimated nonetheless simply as a way of comparing the impact given different soil concentrations for the four time periods.

This assessment will focus on dioxin TEQs. Modeling 17 dioxin congeners provides the most accurate prediction of exposure media concentrations. An alternate is to model a TEQ air concentration as though it were a single compound with a unique set of parameters. Most often, the parameters that have been used are those specific to 2,3,7,8-TCDD. However, this strategy will lead to an inaccurate prediction of exposure media concentrations because a TEQ concentration is, in fact, comprised of 17 congeners, 16 of which have different fate characteristics than 2,3,7,8-TCDD. A unique procedure was developed for assigning fate parameters to TEQs. This procedure is based on a proportional weighting scheme and a representative ambient air profile of dioxin congeners. For example, if 10% of a TEQ air concentration is 2,3,7,8-TCDD, then 10% of a given fate parameter is explained by the 2,3,7,8-TCDD fate parameter. The representative ambient air profile used for this TEQ parameter assignment exercise was generated for the dioxin exposure reassessment document (1) for the purpose of estimating background inhalation impacts.

The general equation used to estimate potential dose normalized over bodyweight and lifetime is as follows:

$$\text{Lifetime Average Daily Dose (LADD)} = \frac{(\text{exposure media concentration} \times \text{contact rate} \times \text{contact fraction} \times \text{exposure duration})}{(\text{body weight} \times \text{lifetime})} \qquad \textbf{(2)}$$

The general equation for estimating excess cancer risk is:

$$\text{RISK} = 1 - \exp(-q^{*}\ \text{LADD}) \qquad \textbf{(3)}$$

where the q^* for TEQs is 0.156 $(ng/kg\text{-}day)^{-1}$. This procedure was used to estimate LADDs and excess cancer risks for all pathways except the mother's milk pathways. In that case, it is not clear that an LADD is meaningful, and all that will be calculated and discussed for the mother's milk pathway is a dose received by the infant in pg TEQ/day.

The exposure parameters used in this screening assessment were developed in the dioxin exposure reassessment document (1). The contact rates include: 20 m^3/day inhalation, 0.2 g/day for a 5-year pattern of childhood soil ingestion, and 104 g/day of above and below ground unprotected vegetables, 100 g/day of whole beef (19% fat), and 300 g/day of whole milk (3.5% fat). The soil ingestion pathway uses the untilled soil concentration rather than the tilled soil concentration; this presumes that the child's contact is not with tilled soils. The vegetable ingestion rate assumes 76 g/day above ground vegetables such as tomatoes, and 28 g/day below ground vegetables, such as potatoes. The total of 104 g/day does not include "protected" vegetables such as peas or corn. Evidence suggests that dioxin impacts only the outer surfaces of vegetation and does not translocate from the roots up the stem and onto leaves of vegetation. The soil dermal contact pattern assumes 350 dermal contact events per year to a body surface area of 1000 cm^2. This body area corresponds roughly to the area of hands, neck, and face. This frequency and surface area contact was crafted to be representative of a farming scenario. As such, the soil concentration term used for soil dermal contact is the tilled soil concentration. Since the mixing depth for tilled soil is 20 cm and for untilled soils is 1 cm, the soil concentrations used for soil dermal contact are 1/20 the concentrations used for childhood soil ingestion. The adherence of soil during each event is 1 mg/cm^2-event, and the absorption fraction is assumed to be 0.03 (3% absorbed). The procedure for estimating the mother's milk dioxin concentration is described in the dioxin exposure reassessment document (1) and taken from Smith (8). The concentration is a function of the mother's intake of TEQs, the half-life in the mother (assumed to be 7 years), the proportion of ingested dioxin that is stored in fat (0.9), and the proportion of mother's weight that is fat (0.3). For this assessment, the mother's intake of TEQs was assumed to be that from ingestion of beef and milk. The infant's dose is calculated as a function of this milk fat concentration, the fraction of milk that is fat (0.04), and an ingestion rate of breast milk (0.8 kg/day). Other exposure assumptions include an exposure duration, lifetime, and body weight of 70 years, 70 years, and 70 kg, respectively. The exposure duration and body weight for children for the soil ingestion scenario are 5 years and 16 kg, respectively.

Table 1 shows all model parameters. Once all the exposures and subsequent cancer risks are estimated, the results will be further examined in terms of the incremental impacts of the four periods, and comparisons to background exposures and risk to TEQs. It is noted that the discussion of the results does not include interpretations of health impacts.

RESULTS AND DISCUSSION

The results of this screening level exposure/risk assessment are summarized in Tables 2 through 4 and Figure 3. It should be remembered that this modeling exercise reflects incremental impacts due to the incinerator only - actual field values at this site may be higher than the modeled values since they reflect impacts from all sources.

Table 2 shows the concentrations in the media during each time period due to the incinerator. The next-to-last column of this table shows the 70-year average concentrations. The final column on this table shows the background concentrations of these media as determined from monitoring data and described in the dioxin exposure reassessment document (1). All monitoring studies available were carefully reviewed, and only those reporting that the concentrations were typical of background, and not known to be impacted by specific nearby point sources, were included. Brief notes on the background media concentrations and the predicted concentrations are:

- **Air**: A total of 84 air samples from urban and suburban settings were averaged to generate an air concentration of 0.095 pg TEQ/m^3 (1), which was rounded to 0.10 pg TEQ/m^3 in Table 2. All these air samples were specifically taken to measure ambient conditions in the urban or suburban settings they came from. Studies which identified a nearby potential source of dioxin release were not included in the inventory. Very few air samples were available from rural settings. However, of those studies available which listed both urban/suburban air concentrations along with rural air concentrations, it was noted that rural air concentrations were 4-6 times lower than urban air concentrations. Hence, the range of 0.02-0.10 pg TEQ/m^3 in Table 2 denotes background air concentrations in rural to urban settings. The annual average air concentration for the exposure setting was 0.11 pg TEQ/m^3 during Period 1, which is similar to urban air concentrations but over 5 times higher than the 0.02 pg TEQ/m^3 air concentrations speculated to occur over rural areas in the U.S.
- **Soil**: Soil concentrations in rural settings in both the United States and Europe showed TEQ concentrations less than 10 ppt (1). The average of data from the U.S. indicated an 8 ppt concentration. The predicted untilled soil concentrations of TEQs approach this background concentration of 8 ppt during Period 2. As discussed below, it is speculated that the model may be underestimating soil concentrations, and hence the soil related exposure pathways of soil ingestion and soil dermal contact. Underpredicting soil concentrations will not greatly affect beef and milk calculations, since soil is

assumed to be only a small part of the cattle diet.

- **Vegetables**: Essentially no data could be found on vegetable concentrations in background settings; the few data that does exist generally shows non-detects with detection limits usually greater than 0.1 ppt (1). Much data, not relevant to the modeling of terrestrial plant impacts, exists on concentrations in potted experimental plants. TEQ concentrations in grasses grown outdoors have been found around the sub-ppt level. Because vegetables are bulky plants and sometimes protected from air-to-plant impacts, it is speculated that background vegetable concentrations are below 0.1 ppt. Therefore, the highest concentration of vegetables modeled at 0.04 ppt of Period 1 appears consistent with available information.
- **Beef**: A total of 14 beef samples were available from grocery store shelves for evaluation and inclusion in the dioxin exposure document (1). The average of these samples was 0.48 ppt TEQ whole beef (19% fat assumed when the literature did not give beef fat levels) when non-detects were assumed to equal ½ the detection limit and 0.29 ppt when non-detects were assumed to equal 0.0. Since the publication of this draft exposure document, a national EPA/USDA beef survey has been completed and was reported on at Kyoto, Japan in November of 1994 at the 14th International Symposium on Chlorinated Dioxins and Related Compounds. A summary of that survey can be found in Winters, et al (9) and a full report on it is being prepared by EPA. From 65 samples of cattle back fat statistically drawn from slaughterhouses nationally, it was found that the lipid concentrations of TEQs, when non-detects are assumed to be one-half detection limit, was 0.89 ppt, and when non-detects are assumed to be zero, the average concentration was 0.35 ppt. Assuming 19% fat for consumed beef, whole beef concentrations from this survey are estimated at between 0.07 (calculated as 0.35 at non-detects equal zero times 0.19) and 0.17 ppt (0.89 * 0.19). The reported range of 0.07-0.48 ppt in Table 2 is comprised, therefore, of the lower value from the EPA/USDA survey and the upper value from the grocery store sample. The predicted concentration of whole beef is higher than these measurements at 4.71 ppt for Period 1 and 1.42 ppt for Period 2. These results are discussed further below.
- **Milk**: Only a very small number of samples of milk could be found which were analyzed for the suite of dioxin-like compounds. The dioxin exposure document lists the background milk concentration at 0.07 ppt TEQ whole milk (3.5% fat), which was calculated assuming non-detects equal one-half the listed detection limits.. Like beef, the Period 1 predicted concentration of 0.64 ppt is higher than this estimate based on limited observations. This is also discussed below.
- **Mother's milk**: The summary of mother's milk data described in the dioxin exposure document indicates a range of 10-30 ng TEQ/kg in milk fat. During Periods 1 and 2, predicted mother's milk concentration is higher at 105 and 31 ng/kg fat.

Air, soil, and vegetable concentrations appear to be generally consistent with background concentrations during periods 1 and 2, and much lower during periods 3 and 4. However, beef, milk, and mother's milk are substantially higher than background during periods 1 and 2, and then lower during periods 3 and 4.

The fact that air, soil, and vegetable concentrations are comparable to background during periods 1 and 2, but beef, milk, and mother's milk are substantially higher than background during periods 1 and 2, is not inconsistent. For vegetables, it was noted above that very sparse data suggests that vegetable concentrations are likely to be less than the typical detection limit of 0.1 ppt, leading to a TEQ concentration less than 0.1 ppt, which supports the modeled value of 0.04 ppt TEQ for Period 1. For soil, two factors are important. One, soil is only a small part of the diet of cattle raised for beef and milk, so high or low soil concentrations would not drive beef or milk concentrations. Two, analysis in the dioxin document suggests that the algorithm to estimate soil concentrations from air concentrations may be underestimating soil concentrations by a factor of 10 or less. Three possible reasons for this underestimation offered were: 1) detritus (die-back) of plant material into soil was not considered, 2) vapor-phase dioxin impact to soils were not considered, and 3) the dissipation half-life of 10 years might be low. This underestimation would impact the soil ingestion and soil dermal contact pathways; i.e., actual exposures and risks due to the Columbus WTE may be as much as ten times higher than reported on Table 3.

While the air concentration of 0.11 pg TEQ/m^3 in Period 1 appears near the reported background range of 0.02-0.10 pg TEQ/m^3, it does not follow that the predicted beef concentration should be near the background range of 0.07-0.48 ng TEQ/kg. First, beef concentrations are expected to be correlated to air concentrations in a rural setting where beef cattle are raised, not correlated to urban air concentrations. The 0.02 pg TEQ/m^3 concentration was developed in the dioxin document as an estimate of TEQ concentrations in a rural setting, whereas the urban air concentration was estimated at 0.10 pg TEQ/m^3, the upper end of this 0.02-0.10 pg TEQ/m^3 range. Therefore, the 0.07-0.48 ng TEQ/kg concentration range in whole beef is expected to be correlated to an air concentration in the 0.02 pg TEQ/m^3 range. The modeled air concentration at the exposure site of 0.11 pg TEQ/m^3 is already over 5 times higher than the expected rural air concentration of 0.02 pg TEQ/m^3.

A validation exercise was conducted for the dioxin exposure reassessment document (1; also published in Lorber, et al, (10)) which showed that the beef concentration is predicted to be about 0.72 ng TEQ/kg starting with an air concentration of 0.019 pg TEQ/m^3, and using all the parameters shown in Table 1 of this exercise. Therefore, an air concentration of 0.11 pg TEQ/m^3 should lead to a prediction that is 5.8 times higher (i.e., 0.11/0.019) than 0.72 ng/kg, or 4.2 ppt. The beef concentration listed in Table 1 is 4.7 ppt for Period 1, which was derived using the air concentration of 0.11 pg TEQ/m^3. The reason it is not 4.2 ppt as predicted by this linear extrapolation is that parameters were developed for TEQs as though this mixture was a single chemical. The validation exercise in the dioxin document separately modeled 17 congeners. The parameters used to model TEQs will not precisely duplicate all exposure media concentrations as compared to a procedure where all 17 congeners are modeled. The result here is that use of the TEQ parameters led to a slightly higher prediction of beef concentration.

The dioxin document also discusses the importance of the beef feedlot fattening regime practiced for most cattle slaughtered for consumption of beef in the United States. It is expected that this 0.07-0.48 ng TEQ/kg concentration range reflects feedlot fattening practices, since the actual beef samples reported in the literature were taken from grocery store shelves, and the EPA/USDA study took samples from slaughterhouses. Beef from grocery store shelves originate from federally inspected slaughterhouses, and the cattle slaughtered at these federal establishments largely originate from feedlots. Only a small percentage of beef consumed nationally originate from cattle which are not fattened in a feedlot. Although home producers of beef may fatten their cattle prior to slaughter, the dilution of residues is likely not be as great as compared to feedlot fattening practices. Model predictions in this screening analysis, and the model predictions of background conditions leading to a concentration of 0.72 ppt discussed above, do not include the impact of feedlot fattening. In the validation of the air-to-beef model described in the dioxin exposure document, a final prediction of 0.36 ppt whole beef included a 50% reduction due to feedlot fattening. This was based on published modeling studies which suggest that this regime reduces concentrations by 50% of what they are prior to the entry into this feedlot (5, 11).

In summary, the high beef concentration of 4.71 ppt in Period 1 does not appear inconsistent with available data, considering that the air concentration driving this prediction is over 5 times higher than expected in a rural setting, and the dilution effect of feedlot fattening was not considered.

The TEQ concentration predicted for milk in Periods 1 and 2, 0.64 and 0.19, respectively, are higher than background because air concentrations predicted to occur at the Grade A Dairy farms are higher than what are expected to occur in a rural setting. Like beef, therefore, these predictions do not appear inconsistent with available data.

Modeled 70-year average concentrations were used to determine the 70-year exposures and risks that are shown in Table 3. As shown in this table, the vegetable, beef, and milk pathways all exceed the inhalation pathway, with beef and milk exposures exceeding inhalation exposures by about two orders of magnitude. A lifetime of soil dermal contact associated with farming activities appears to lead to an exposure about two orders of magnitude lower than the inhalation pathway. The childhood soil ingestion pathway is not a 70-year exposure pathway, but rather a 5-year exposure pathway. For each period, the untilled soil concentration was used to estimate what the soil ingestion exposure and risk would be during that period. Results suggest that the childhood soil ingestion pathway is comparable to the inhalation pathway.

Table 4 shows the daily dose via beef and milk ingestion, and the daily dose to the infant from breast milk. These doses are compared to background doses. The background doses were derived in the dioxin exposure document (1) and are a function of background concentrations and average contact rates for the various pathways. For beef and milk ingestion, the background dose assumes: 77 g/day beef ingestion at 0.48 ng/kg and 251 g/day milk ingestion at 0.07 ng/kg. The beef and milk doses due to the Columbus incinerator are calculated assuming the lower ingestion amounts of 77 g/day beef and 251 g/day milk. This was done so that comparison to background doses as calculated in the dioxin exposure document is more equitable. As seen, the dose calculated for Periods 1 and 2 exceed the background dose by about 12 and 4 times, respectively.

Mother's milk dioxin concentration is assumed to be directly a function of the mother's intake. For this assessment, mother's intake (attributable to the incinerator) is characterized by the sum of beef and milk intakes. For period 1, mother's intake was assumed to be 663 pg TEQ/day, and using the methodology for estimating infant dose, a dose of 3020 pg TEQ/day was calculated. In contrast, the dioxin document calculates a background dose for infants of 600 pg TEQ/day. This assumes a mother' milk dioxin concentration of 20 ppt (based on data and verified by modeling mother's milk fat using background doses of dioxin; see EPA (1) for further details), which is 1/5 of the 103 ppt TEQ in mother's milk which is predicted to occur from a mother's intake of 663 pg TEQ/day. For the second period, the prediction of 30 ppt in mother's milk is 50% higher than the 20 ppt background assumption, leading to an infant dose of 906 pg/day, also 50% higher than the 600 pg/day background dose.

Figure 3 shows the increments of the 70-year exposures which occur for each pathway during each time period. For the vegetable, beef, and milk ingestion pathways, and for the inhalation pathway, over 90% of the exposure is the result of emissions which occurred during Period 1, or from 1982 to mid-1994. For the dermal contact pathway, however, most of the exposure occurs in Period 3. This is because the dioxins have built up in the soil over time, so that the 30 years during period 3 contains significant residues that have built up from periods 1 and 2.

By this analysis, most of the exposures for the ingestion and inhalation pathways occurred during the first period. The annual exposure and risk during the second time period was estimated to be only about 1/3 to 1/4 of what it was in the first period. The annual risk can be thought of as the risk incurred by an individual that moved into the area for one year only. The reason that the annual risks of periods 1 and 2 do not vary significantly is that the exposure media concentrations are not that much different. This can be seen from the results given in Table 1. The reason that the percentage of total lifetime risk is so much higher in Period 1 as compared to Period 2 (as seen in Figure 3) is that the annual risk for Period 1 was experienced for 12.5 years, whereas the lower risk of period 2 was experienced for only 2.5 years. Once the MACT is in place and emissions in period 3 are 1/100 of what they were in period 2, exposures and risks are significantly lower.

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Table 1. Model parameters for screening assessment. Nomenclature follows that in EPA (1); see that reference for a full description of models and parameters.

Name	Description	Value
	I. Particle Depositions	
kw	first-order weathering constant, yr^{-1}	18.01
ks	first-order soil dissipation constant, yr^{-1}	0.0693
Yg	yield of grass, kg/m^2 dry	0.15
Ig	interception of grass, fraction	0.35
Yh/s	yield of hay/silage/grain, kg/m^2 dry	0.63
Ih/s	interception of hay/silage/grain, fraction	0.62
Yveg	yield of above ground vegetables, kg/m^2 fresh	7.8
Iveg	interception of above ground vegetables, fraction	0.48
Vd	dry deposition velocity of particles, m/sec	0.002
M	mass of mixing soil, kg/m^2	10
Rw	wet deposition retention on plants, fraction	0.30
	II. Vapor Transfers	
VGgr	empirical correction factor for grass, unitless	1.00
VGh/g	empirical correction factor for hay/silage/grain, unitless	0.50
VGveg	empirical correction factor for above ground vegetables, unitless	0.01
	III. Below Ground Vegetables	
Dtill	depth of tillage, cm	20
OCsl	soil organic carbon, fraction	0.02
VGbg	empirical correction factor for below ground vegetables, unitless	0.01
	IV. Bioconcentration	
Bs	bioavailability on soil relative to vegetation, unitless	0.65
DFbs	beef cattle soil diet fraction	0.04
DFbg	beef cattle grass diet fraction	0.48
DFbh/s	beef cattle hay/silage/grain diet fraction	0.48
DFds	dairy cattle soil diet fraction	0.02

Table 1 (cont'd).

Name	Description	Value
DFdg	dairy cattle grass diet fraction	0.08
DFdh/s	dairy cattle hay/silage/grain diet fraction	0.90
V. Fate Parameters Specific to TEQs		
VAP	vapor fraction (sorbed fraction = 1 - vapor fraction)	0.27
Bvpa	air-to-leaf transfer factor, [ng TEQ/kg plant dry]/[ng TEQ/kg air]	$4.55*10^{5}$
log Kow	log octanol water partition coefficient, unitless	7.04
H	Henry's Constant, atm-m^3/mole	$9.16*10^{-6}$
RCF	root concen. factor, [ng TEQ/kg plant fresh]/[ng TEQ/kg H_2O]	$1.21*10^{4}$
Koc	organic carbon partition coefficient, L/kg	$1.42*10^{7}$
VI. Exposure Parameters		
IR	inhalation rate, m^3/day	20
SI	childhood soil ingestion rate, g/day	0.2
BMI	infant breast milk ingestion rate, g/day	800
VI	vegetable ingestion rate, g whole/day	104
BI	beef ingestion rate, g whole/day	10
MI	milk ingestion rate, g whole/day	300
CF	contact fraction for soil, vegetable, beef, and milk ingestion	1.00
DE	number of dermal contact events per year	350
A	body area over which dermal contact occurs, cm^2	1000
AD	adherence of soil during contact event, mg/cm^2-day	1.0
DA	fraction of dermal contact which is absorbed	0.03
LT	lifetime, yrs	70
BW	adult body weight, kg	70
EDc	exposure duration for childhood soil ingestion, yr	5
BWc	body weight for children for soil ingestion, kg	16
EDi	exposure duration for infant breast milk ingestion pathway, yr	1
BWi	body weight for infant for breast milk ingestion, kg	10

Table 2. Summary of exposure media concentrations for TEQs for the four periods and averaged over a 70 year period.

Exposure Media	T_1, yr	Conc	T_2, yr	Conc	T_3, yr	Conc	T_4, yr	Conc	C_{avg}	C_{bkgrnd}
Air, pg/m^3	12.5	0.11	2.5	0.03	30	0.0003	25	$7*10^{-6}$	0.02	0.02-0.10
Soil, untilled, ng/kg	12.5	3.9	2.5	7.6	30	4.1	25	0.5	2.9	8.0
Soil, tilled, ng/kg	12.5	0.2	2.5	0.4	30	0.2	25	0.03	0.15	8.0
Vegetables, ng/kg fresh	12.5	0.04	2.5	0.01	30	0.0001	25	$1*10^{-5}$	0.008	<0.1
Beef, ng/kg whole	12.5	4.71	2.5	1.42	30	0.07	25	0.01	0.92	0.07-0.48
Milk, ng/L whole	12.5	0.64	2.5	0.19	30	0.007	25	0.001	0.12	0.07
Mother's milk, ng/kg fat	----	105	---	31	---	1	---	0.2	---	10-30

Notes:				
	Period 1, T1:	1/1/82 - 6/31/94	-	incinerator operation before combustion improvements
	Period 2, T2:	7/1/94 - 12/31/96	-	incineration operation after combustion improvements but before MACT technologies
	Period 3, T3:	1/1/97 - 12/31/26	-	incineration operation after MACT installation
	Period 4, T4:	1/1/27 - 12/31/51	-	incinerator no longer in operation until 70 year lifetime

Media concentration notes:

1) air includes vapor plus particle phases, 2) untilled soil concentrations used for soil ingestion pathway, tilled soil concentrations used for dermal contact pathway, and 3) beef assumes 19% beef fat; milk assumes 3.5% milk fat, 4) C_{avg} is the 70-year average concentration, and C_{bkgrnd} is the background concentrations as developed in the dioxin exposure document (3), 5) For mother's milk calculation, the number of years in each period is not relevant since the exposures are for one year and infant's dose to mother's milk is estimated for each period.

Table 3. Exposure and risk results.

Exposure Pathway		Lifetime Average Daily Dose, ng/kg-day	Excess Cancer Risk
Soil Dermal Contact		$6*10^{-8}$	$9*10^{-9}$
Vegetable Ingestion		$1*10^{-5}$	$2*10^{-6}$
Inhalation		$6*10^{-6}$	$9*10^{-7}$
Beef Ingestion		$1*10^{-3}$	$2*10^{-4}$
Milk Ingestion		$5*10^{-4}$	$8*10^{-5}$
Soil Ingestion	Period 1	$3*10^{-6}$	$5*10^{-7}$
	Period 2	$7*10^{-6}$	$1*10^{-6}$
	Period 3	$4*10^{-6}$	$6*10^{-7}$
	Period 4	$4*10^{-7}$	$7*10^{-8}$

Table 4. Comparison of background dose of TEQs from beef and milk with those predicted to occur for the four time periods.

Pathway	Background dose, pg/day	Period			
		1	2	3	4
Beef and milk ingestion	55	663	199	9	1
Breast milk ingestion	600	3020	906	41	6

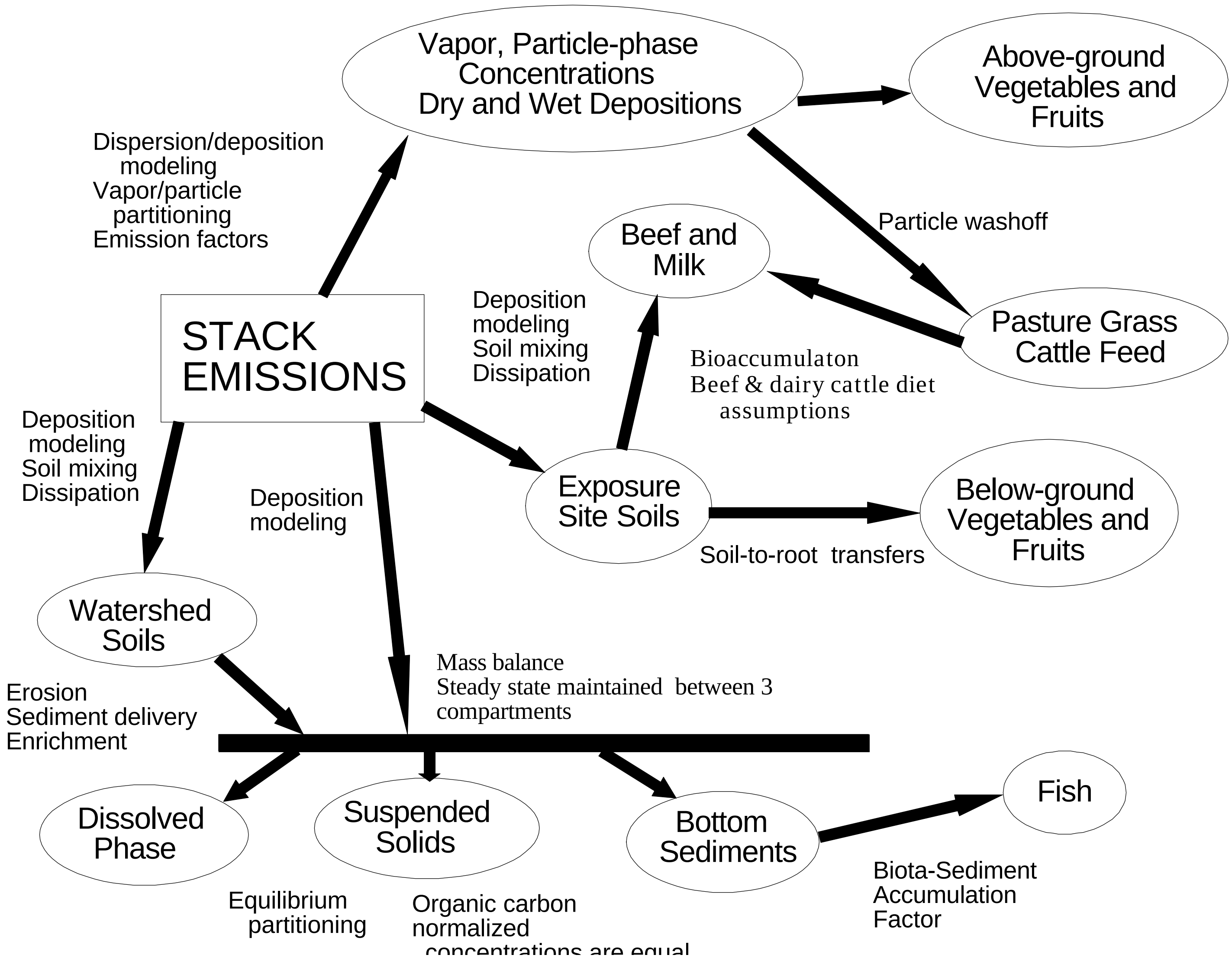

Figure 1. Diagram of the fate, transport, and transfer relationships for stack emission sources (from the draft Dioxin Exposure Document (3)).

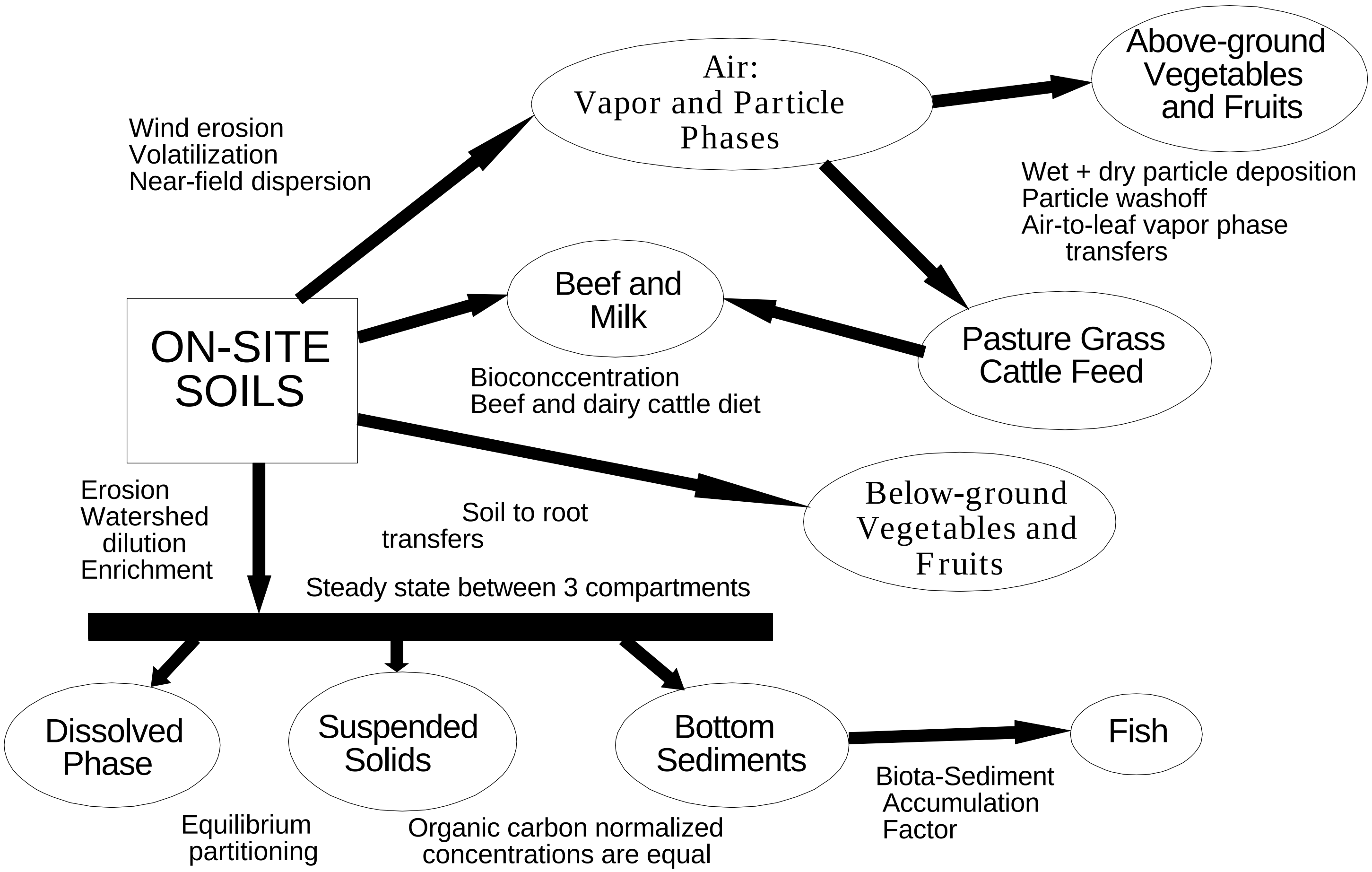

Figure 2. Diagram of the fate, transport, and transfer relationships for soil sources (from the draft Dioxin Exposure Document (3)).

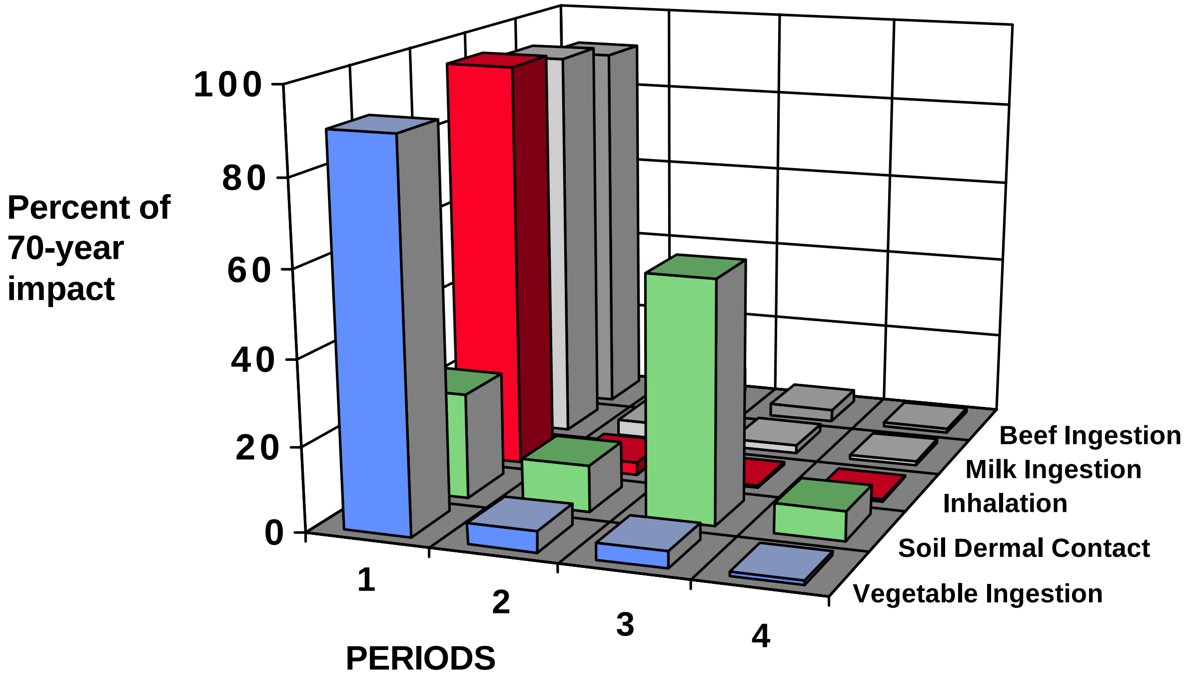

Figure 3. Percent of 70-year exposure and risk which occurs during each of the four periods for five exposure pathways.